NOTES ON A SERIES OF CASES OF SORE THROAT ARISING FROM A MILK-BORNE INFECTION.

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NOTES ON A SERIES OF CASES OF SORE THROAT ARISING FROM MILK-BORNE INFECTION

Milk supply is a subject which has received much consideration and in order that a supply of pure good milk may be obtained much legislation has been enacted, but in spite of all the care with which that legislation has been carried on there are still frequent outbreaks of disease arising from milk-borne infection.

In a great many (nearly all in fact) of those outbreaks of enteric fever and scarlet fever and some other infectious diseases arising in connection with milk supply it is found that the infection has been imparted to the milk from a human source, usually an unrecognised case of the disease.

To deal with such cases we have the various Infectious Diseases Acts and Orders and I do not intend to give such any consideration at present, except in so far as there is a relationship to the outbreak which I am about to describe, and which had as far as could be ascertained no relationship to infection from any human source.

The main characteristic of this epidemic was sore throat which is a very common ailment and which is very prone to occur more or less in epidemic form, the other characteristics were a variety of ailments which have for some time been classed together more or less indefinitely as rheumatic

manifestations and particularly those designated acute rheumatic fever and endocarditis and pericarditis.

These two conditions, acute tonsilitis and acute rheumatism, have been long associated and Dr. Garrod in a lecture at Leeds in 1889, discussed the intimate relationship between them.

He and Lennox Browne have shown that this was recognised by Musgrave in 1710; John Ball in 1762; Sauvage in 1771 and Stoll in 1777. Haig Brown in 1886 states that tonsillar inflammation is sometimes truly rheumatic.

Sir Andrew Clark in 1889 writes thus:
'If one will think merely of the rapidity with which the tonsil manufactures and discharges lymph cells, it will not be difficult to see how a sudden suppression of this process, the accumulation of effete matters in the crypts and the filling up of the lymph spaces with products of bacterial life, and matters undergoing evolution, may contaminate the blood and originate troubles considered as rheumatic.'

Lennox Browne accepted this view of Sir Andrew Clark but later acknowledged that the blood poisoning was of microbic origin.

Now the intimate connection between the sore throat and rheumatism is very generally acknowledged, and several epidemics of sore throat with rheumatism following have been described and will be referred to later on.

From a study of this outbreak I expect to be able to

still further corroborate the intimate connection between the two diseases or shall we say the two parts of the one main disease and endeavour to associate certain micro-organisms with them. I also intend to review some of the reports of previous epidemics and endeavour to associate the origin of the trouble with disease of the cows' udder and in doing so refer to cases where such disease has been supposed to produce scarlet fever, and make some suggestions as to how these outbreaks may be minimised or abolished altogether.

DESCRIPTION OF CASES

My first indication of an epidemic occurring was on the 17th of May 1911, when I had five new messages and found all of these to be cases of sore throat. Most of them had been taken ill the day previous, i.e. the 16th, and when I saw them they very closely resembled scarlatinal sore throats.

I knew that several of them were supplied from a certain dairy and on enquiry found that the others had received milk from the same source. These cases were all in one locality. On the following day there were many more throughout different parts of the district and every day for about ten days there were fresh cases of sore throat. I did not keep exact records of the number of cases each day, but within 10 days I had prescribed for over a hundred cases. Many of these were not so bad as to be laid up but were able to come to the surgery and many came only after they had been ill for several days and when it had become known that the milk supply was suspected. In many families several members were affected and in most families more than one. Even in those families where only one asked for advice, on enquiry it was found that several others had complained to a less extent. There was only one case where I could find no connection with milk from this dairy, but he may have had an ordinary sore throat as is usually found or

have taken milk in some house that he did not remember.

There are five other medical practices in this district and these had cases which likewise showed that one milk supply was the probable source of infection. The epidemic therefore was considerable in the number of cases.

Cases showed themselves in every district supplied from this dairy, several being in neighbouring villages, but even there the cases were limited to the supply from this one dairy. The dairy had a double source of supply but one of these sent milk also to another district and on enquiring there, I found no evidence of prevalence of sore throat, so that it became limited in origin to one farm supply. There are various other dairies in the districts but their customers were practically free from sore throats.

There was no practical doubt of the one supply of milk to the dairy being the source of the infection.

The assistant M.O.H. investigated the outbreak, even although it was not a notifiable disease, about a week after its commencement. He took swabs from various cases and occasionally I sent swabs from others where diphtheria was probable, but there never was any evidence of diphtheria found. Every swab showed a growth of staphylococcus pyogenes aureus.

The assistant M.O.H. visited the farm suspected and ascertained that one milkmaid began to complain on the 15th May and that others concerned in the dairy took ill on the 18th, 19th and 20th May. There were over 40 cows, but

these were healthy except for one cow with a 'weed' and they were not using the milk from the TWO hind teats. The M.O.H. advised that this cow's milk be not used. I subsequently ascertained from the farmer that he was practically the only one at the farm who had not been ill there. Their illnesses were practically the same as those in Cambuslang and as the first milkmaid to become ill sickened only one day before the first cases here while the others at the farm took ill at the same time it was not possible that they could be the means of giving the infection to the milk.

The farmer also remarked to me that he did not think the milk bad at all, although the quarter of the udder was a little hard.

Putting this statement along with the statement to the assistant M.O.H. that the milk from the two hind quarters was not used, I come to the conclusion that the milk was used practically as much as it could be, because I know that no farmer (unless he is up to the times and in advance of them) would ever think of putting aside the milk from the two hind quarters when only one was diseased, that from the diseased quarter only being usually milked on to the ground when it is too thick to mix with the milk for using. I am afraid that in many cases there is not sufficient time allowed for the milk from an inflamed quarter to become free from infection before it is again mixed with the rest

of the milk. This cow was not newly calved but was nearing the end of her lactation period and to all appearance became all right again but was sold and so lost trace of.

At the other farm from which milk was supplied to the dairy in question, M.O.H. found that several cases of scarlet fever had occurred, two being then in hospital, one home a fortnight previously and one two weeks earlier. It is probably noteworthy that these cases were found as three cases of scarlet fever occurred in Cambuslang at this time with no known source of infection, they used this milk and one of them had an exceptionally bad throat as if she had the infection of scarlet fever and of this other epidemic as well, it may be that the latter predisposed to the former. Strange to say a small outbreak of scarlatina occurred in a neighbouring district several months later and all were supplied with milk from the latter farm, but there were then fresh cases of scarlet fever there.

I shall now consider 'weed ,' 'garget' or inflamed udder, (acute mammitis.)

This very often occurs shortly after calving but some of the worst and most severe on the cow occur near the time when milking is about over for the season or even when the cow has ceased to give milk.

It is usually attributed to a chill and undoubtedly a chill is often the exciting cause but like other acute

inflammations it is now known to be microbic and both staphylococci and streptococci are usually present but the exact relationships are not decided.

The first symptoms discovered may be, not the hardness of the vessel but the presence of a little clot or two of milk in the strainer, sometimes there may be a little blood. In a mild case a cow may recover with very few signs and symptoms, and an ordinary milker may entirely fail to recognise any illness.

In the usual form of weed, however, the quarter shows quite a hard swelling, the milk becomes watery and yellow and less in quantity and often clots of milk are present. This may continue for several days and sometimes the garget runs on for a considerable time even until after the cow has ceased to give milk and the quarter may remain permanently affected and give little or no milk afterwards. In other cases there is evidence of acute systemic disturbance with high fever as well as the local inflammatory condition.

There is no doubt of some of these cases being very infectious and sometimes quite a large number of cows are infected in a comparatively short time and in these cases it is not uncommon to find that the udder shows very little although the cow may be very ill.

In other cases where the infection is not so virulent it may show its infectivity by several cows on one side of a byre being affected while there are none on the other side. Not uncommonly one finds a cow or two near a

a diseased cow with scabs and ulcers on the teats as if it was a local infection from the fingers of the milkers which is the usual method of the infection spreading in a byre.

In a dairy of cows well kept and cared for, I found this local infection, the cow next to one with a sub-acute weed having scabs on the teats. In this dairy I found two cows with swollen joints which began after they had a weed which caused them to be very ill for several days. They were apparently rheumatic joints resulting from udder infection, this is, in my opinion, further corroboration of my view to be stated later on.

When a cow is found to have an inflamed udder, the usual practice is to refrain from using the milk from the diseased quarter so long as it shows visible evidence of the milk being distinctly bad.

If it is a farm where calves are reared, then it is the usual practice to give the milk from that cow to feed the calves if milk is required, and although I have carefully enquired, I have never found evidence of calves being the worse for such milk.

Where the milk cannot be used in this way it is commonly milked on to the ground under the cow (rarely into a vessel and taken away) so that there is every chance of the cow lying down and the other teats being covered with the germs as well. Again, the cow may be at any part of the milking, and no milker used to think it worth while to

cleanse the hands after milking such a cow, but commenced to milk the next cow with at least one hand covered with infective material. This was the common practice at the dairy concerned with the outbreak under consideration, but now the milkers are expected to wash their hands in weak lysol solution after milking each cow.

I am afraid, however, that too many are in the habit of selling in the general milk all that can be milked from a diseased cow, provided the milk is not clotted.

It seems, therefore, a certainty that milk is often infected with the germs which cause acute mammitis in the cow and that may occur in some cases in spite of the utmost care the dairyman may take as the germs may be found even before the udder shows disease. In this respect reference may be made to a report in the Journal of Infectious Diseases, January 1912, Chicago, in which H.E.Ross records that during a series of experiments (counting the cell contents of milk) it was found that the cell content from one cow became abnormally large and this was found on examination to be due to a garget in one quarter from which the cells were nearly 62½ millions per cc. while from the others there were less than ½ million.

Even although one went to the trouble of bacteriological tests and leucocyte counts for the examination of the milk, it would not be always easy to detect infection because as is shown in the Journal of Chemical

Research, January 1912, there are always streptococci and leucocytes present in the milk of healthy cows and any standard to be in all cases effectual to exclude disease would exclude a great deal of milk from perfectly healthy udders, and again, it is as yet impossible to differentiate between the pathogenic and the non-pathogenic streptococci.

When it is considered also how many cows would have to be examined practically daily to be of any efficiency for the prevention of infection, that method is not practical.

If only the mixed milk from each dairy was examined, then the dilution would be so great that from a count the inference would not be reliable, but a high leucocyte count would undoubtedly be suspicious.

CLINICAL FEATURES OF THE CASES

The first and most frequent symptom was the sore throat.

The typical moderately severe case usually commenced rather suddenly with shivering, feverishness, temperature rising quickly to 103°, no vomiting but severe headache, backache and general soreness. The throat became painful and showed intense redness all over the pharynx and soft palate, the tonsils became swollen and each lacuna seemed filled with a yellow plug. These plugs gradually increased in size and spread peripherally till a yellowish or creamy patch appeared by their coalescence. This continued for several days, then the temperature diminished, the pains and aches disappeared and in about five days the patient was walking about fairly well, but very pale and feeling very weak. The lymphatic glands were usually much swollen and very painful and remained hard and tender for some time after the throat was better.

That was the average, but there were many variations. As I have previously stated, many cases were so slightly affected that they did not cease work and their throats showed symptoms of mild lacunar tonsillitis. In other cases there was a much more severe tonsillar affection - the tonsils sometimes became so swollen that they met in the middle line, the uvula being at the same time much swollen and oedematous. In a few cases, a tonsil became

very swollen & an abscess formed and burst if not evacuated and in at least two cases first one tonsil suppurated and then the other. These may be called peritonsillitis but in my opinion peritonsillitis is practically always an abscess in the tonsil extending outwards and upwards, and I think this view is corroborated by the fact that these abscesses are best treated by a pair of sinus forceps inserted at the large lacuna seen near the upper part of the tonsil.

I was repeatedly surprised to find cases of tonsillitis recovering, to the patient's mind, completely and as far as appearance and pulse went, normal, yet showing by the thermometer a temperature of 100° for several days longer.

In some cases the throat was so intensely congested that little vesicles appeared over the soft palate, in other cases there was a general redness all over the tonsil and pharynx with very little swelling and very little of the lacunar deposit and no false membrane.

The tongue was usually considerably furred, but did not give the strawberry appearance of scarlet fever at first, although sometimes it was not unlike the red strawberry tongue afterwards.

A great many of the cases recovered without further illness, but almost invariably they were pale and weak for a week or two even after all throat symptoms had disappeared.

The most frequent sequela was acute rheumatism, this usually commenced about nine or ten days after the sore throat, but was sometimes earlier or later. Several of the cases were very severe and showed all the symptoms of acute articular rheumatism and affected many joints large and small. One case, a woman over 40 years of age, was very pale and anaemic and had not been in good health for a long time, had a moderately sore throat (her husband and son had both a similar throat but were not further affected) In about a week she began to complain of pains in her joints and both arms and legs became greatly affected. Practically every joint in these became swollen and painful. For a month she was scarcely able to move a limb. Near several of the joints subcutaneous nodes appeared. Gradually the joints became less painful and in three months she was able to be sent away for a change to a convalescent home. She was not, however, quite back to her normal even six months after the illness commenced, but appeared to be likely to recover entirely. In this case there were no other complications, no heart disturbance ever showed itself.

Another woman, also about 45 years of age, had taken a chill about a month before this outbreak which was considered to be influenza and then developed acute rheumatism from which she was suffering to a slight extent when the epidemic of sore throats commenced and from which she suffered very slightly, but the rheumatic condition became

very much aggravated a week later and the joints became much more involved and some which had nearly recovered became even worse affected than at first. In this case although the heart never gave any evidence of valvular disease, it was extremely rapid and feeble, so much so that I was at times afraid to give as much salicylate as I would have liked. The sweating in this case was very profuse.

Every joint in the upper limbs and all except the hips in the lower limbs were very markedly swollen and some of them very red. As the swelling subsided considerable thickening was left especially noticeable about the wrists. Although her illness commenced in April and was aggravated by this attack near the end of May, it was the last week in August, i.e. three months later, before she could be taken away for a change and even then she could not walk for any distance, not even to the railway station. She improved considerably during her month's stay at the coast and when she came home, she could move about the house and do a little household work, but the ankles and insteps were still swollen and painful, as were also the wrists and some of the joints of the fingers and even the other joints of the arms seemed to be still affected although there was little to be seen about them as she was unable to get her hands up to do her hair and such like movements. I saw her as late as January, i.e. 8 months after the epidemic and

still there was some stiffness and swelling about the wrists, but the feet were very well although she still wore cloth boots.

These were the two most severe cases of rheumatism in that form but there were many others which may be considered rheumatic, in which there was not so much disturbance, in fact, several of those who had had sore throat complained of pains in their joints but would not go off work and managed to get rid of their pains without so doing.

Others were off duty for only a few days being very quickly relieved by salicylate and getting out to work as soon as the pains were away and sometimes they had no return. It is very difficult to get some people to lie in bed and refrain from working when they feel nothing the matter with them and not a few learned in the bitter school of experience that it is not wise to return to work immediately after even a very mild attack of rheumatism, affecting even but one or two joints and had to endure several weeks in bed with an attack of rheumatism very much worse than the original attack, as if the disease was very liable to re-assert itself whenever conditions were suitable.

In many cases there was a history of indefinite aches and pains in the joints or muscles which are popularly called rheumatic, but which the scientific person has been tending to exclude from coming under the term rheumatic, and these aches seemed to recur for months when the person

was exposed to a wetting or a chill.

I consider that in all these cases the term rheumatic was strictly applicable as they apparently originated from the same source as the real rheumatic fever.

Another condition which came on after the sore throat was heart trouble. I have said that neither of these cases of rheumatism which I have described had any gross heart disease, although it appeared as if the second case had considerable myocarditis but several others were not so fortunate.

The most tedious and anxious case was a lady of 28 years of age, married for five years but no family. There was no history of any previous illness likely to cause heart trouble and up till a month before I saw her she had enjoyed good health. Her husband had attended me for his sore throat and weakness following it at the time of the epidemic, but the lady's throat was only slightly affected and as her husband was a chemist he suggested her taking some of the medicine I had prescribed for him and I did not see her then. She became gradually worse, however, and I was asked to see her nearly four weeks after the epidemic commenced.

When I saw her first her heart was extremely rapid practically uncountable and on several occasions she had apparently become almost unconscious after sitting up or moving in bed.

The only joints which were affected in her case were the wrists which were swollen slightly and painful, and which became quite normal in less than a week.

This lady was extremely nervous and excitable and I thought that part of the rapidity of the pulse was caused thereby. She was in quite a terror that something was going to happen. With rest in bed the condition improved and I could discern what I considered was a pericarditis as well as some endocarditis. After several weeks she became anxious to get up and tried to get to a couch with the result that the heart condition became much worse. I called in a consulting physician with a very wide experience and his opinion was similar, i.e. that there was both pericarditis and endocarditis. She was kept in bed or on the couch for three months and then was sent off for a holiday to her own family where she remained for another two months doing practically nothing. After she returned, I examined the heart again and found a considerable improvement in the condition of the heart muscle as it was working more easily and to better purpose. I could not detect pericarditis but the endocardial murmur was still evident. Otherwise she was feeling very well and able to do a little housework and move about but was breathless if she exerted herself to any extent and could not endure excitement on account of it causing palpitation.

Another case, a boy of ten years, had the usual sore

throat with acute rheumatism following, which was quickly recovered from and I had ceased to attend him over a week when there was a fresh complaint that his legs were very stiff and he was very weak. He now showed a return of the rheumatism in the hips but in addition his heart was much dilated and gave sounds which might mean either endocarditis or simply dilatation.

The temperature was very little and soon became normal when the lad was kept in bed. Even although the heart resumed almost its normal size, the mitral systolic murmur became more pronounced, and is still present and will almost certainly be permanent.

There were only two cases that showed to me distinct evidence of pericardial friction, but from the way in which I have known a distinct pericardial rub to disappear in a night, I consider that other cases which gave no pericardial friction but showed extreme heart weakness, breathlessness and palpitation may have had some pericardial inflammation.

Of endocarditis there were not many examples in adults but quite a few children who had no rheumatism at all and even very little sore throat were under treatment for this illness.

Other ailments noticed were:- erythema nodosum several cases both adults and children; two of these were affected on both arms and both legs. Purpura haemorrhagica in one boy of 12 years who suffered also from acute

articular rheumatism and endocarditis. This poor lad was unfortunate enough to acquire syphilis about five years previously and this may have had some effect in making his condition worse. He is still, 8 months afterwards, unable to run about and play footbal as his joints are so stiff. He had a recurrence of the rheumatic fever about six months after the original attack and there was still a mitral murmur present.

There were no cases of chorea at the time of the epidemic but three of those who had sore throat have since suffered from this disease.

The foregoing are the chief illnesses which I associated with this outbreak. Whether any of the pulmonary conditions which occurred were so associated I cannot say. At the time of the outbreak I did not consider these cases of pneumonia or broncho-pneumonia to be so caused, but I remember that there were some which developed after the sore throat.

There were no cases of general septicaemia or of death.

After the epidemic for some time I frequently had people coming to the surgery complaining of pains in the limbs and headache as if the disease was still in the system and liable to recur if the person was exposed to a chill.

I will now consider the connection between the sore throat and the rheumatism. I have said that it was usually a period of fully one week from the commencement of the sore throat till the commencement of the rheumatic fever as if that was the incubation period of the rheumatic fever and as a further proof of such being the case, I may mention a case which occurred nearly 20 years ago when I was just commencing medical study:- A man was bitten by a house rat rather severely on the hand and this was followed in nine days by a sharp attack of rheumatic fever. The friends blamed the rat bite at the time and I wondered if it was possible, although I had not heard of such a theory being suggested for rheumatic fever.

In all likelihood the same class of germ entered the system from the bite as did so from the sore throat. Acute sore throat has been described in all the books for a long time, but it is not always described in the same way. As a very eminent authority on this condition let reference be made to Herbert Tilley as he describes it in Green's Encyclopoedia or in a clinical lecture published in the Clinical Journal, July 1911. His classification in the former is:

i. Toxic Tonsillitis
   (a) Rheumatic
   (b) Catarrhal or Influenzal
   (c) Tonsillitis in infectious diseases as scarlet fever.

ii. Infectious with Exudate
   (a) Lacunar
   (b) Diphtheritic

iii. Parenchymatous tonsillitis) both of which
iv. Peritonsillitis) may suppurate

v. Phlegmonous or Gangrenous (in septic pharyngitis)

vi. Acute Ulcerative Tonsillitis.

and various other chronic and specific forms which do not concern the present purpose.

In his clinical lecture referred to he simplifies the classification by dividing acute sore throats into three fairly well defined types:-

1. Lacunar
2. Parenchymatous tonsillitis
3. Peritonsillitis which frequently suppurates and is called 'Quinsy'

Later on in his lecture, however, he states that he is not going to mention 'Ulcerative Tonsillitis' nor Vincent's angina nor those due to streptococci or pneumococcic ulceration, his object being only to review the common types of acute and chronic sore throats.

His only reference in the lecture to rheumatic fever is a warning not to give a hurried good prognosis in a case of sore throat in a child under 4 years, lest next day a rash of scarlet fever, measles or symptoms of rheumatic fever appear.

If a return is made to his description of acute tonsillitis in Green's Encyclopoedia, even although he puts it in the forefront in the classification, he makes

practically no suggestion as to the special class that is likely to be rheumatic but describes them all practically under the same description as in the lecture.

Lennox Brown in his text-book on diseases of the throat makes reference to Morell Mackenzie's division, viz:

Angina follicularis which is non infectious &

Angina lacunaris which is typically infectious.

Throughout the whole epidemic here I did not find evidence of an actual infection from one person to another As I have already stated, the climatic conditions were not such as are usually considered likely to give sore throats.

F.T.Roberts states, and it is usually acknowledged to be the case, that a state of ill health from any cause, predisposes to tonsillitis, as also do mental depression and anxiety, but many individuals are attacked when in apparently good health. In the epidemic under review, there were many cases of the latter but there were several who were not in good health who suffered very severely.

Lennox Brown writes that in many cases in which the disease does not reach suppuration, resolution of the local trouble is followed by a smart attack of rheumatism or rheumatic gout.

Dr. T.K.Monro associates the parenchymatous tonsillitis with acute rheumatism but makes no reference to rheumatism

as following other forms of tonsillitis.*

In many descriptions of tonsillitis reference is made to rheumatism following but often no special distinction is made as to what form of sore throat is most likely to be associated with rheumatism.

The description which is most exact is that of Dr. Garrod, viz:- "A diffuse erythematous redness is seen to occupy the whole of the back of the throat, and some oedematous swelling of the mucous membrane is present, which is most marked above the uvula which is swollen and elongated. The pharynx is moist and free from all exudation, one or both tonsils may present more or less considerable swelling."

That is not the same as my description of the average sore throat in this epidemic, but the sore throat intensity did not correspond to the rheumatic condition but rather the opposite. I had no case which went on to suppuration that developed rheumatic fever, and many of the worst sore throats with much exudation and white membrane gave comparatively little general systemic infection, whereas several of the cases of rheumatism never sought advice for the sore throat and did not consider it worth mentioning

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* Dr. Poynton in his article on Rheumatism in Green's Encyclopoedia states that the Angina Faucium is not distinctive but is usually catarrhal and rarely suppurative, but there may be a membranous deposit upon the tonsils or a condition of follicular tonsillitis.

till asked about it. The throat which I usually considered most suspicious and which was apparently the one most frequently associated with the rheumatic condition was one in which there seemed to be a mild lacunar sore throat, the yellowish plugs being comparatively small and few in numbers but the redness of the whole throat, tonsils and fauces and soft palate, very marked and often there was a vesicular eruption on the soft palate and the uvula swollen and oedematous.

E T I O L O G Y

I shall now refer to the etiology of acute tonsillitis and rheumatism. It has been said by some (among them Lennox Brown) that the same general description may apply to these two diseases thus:- an acute infectious disease occurring most frequently about the middle period of life but not often found in persons below 5 years of age nor above 50 years, usually brought on by exposure to cold or damp and especially in those with a hereditary tendency thereto or who are in impaired health or suffering from mental depression or overstrain. One attack predisposes to other attacks and frequently recurring attacks in the same house are often associated with imperfect drainage.

Let us further consider the bacteriology of the two conditions. As to the micro-organisms present in acute tonsillitis, the general agreement is that there is usually a mixed infection. Normally in healthy throats both staphylococci and streptococci are found but not often, if ever, the staphylococcus pyogenes aureus, in the acute tonsillitis this is often found and in every throat examined in this epidemic it was present. In these cases it was usually the membrane which was swabbed for examination on account of the suspicion of diphtheria and probably if the other form of tonsillitis with little of such

exudate had been examined it might have given the streptococcus as the predominant microbe.* In the epidemic at Boston which I quote later, there was no constant organism found in the throat and it may have been that in that outbreak the various classes of sore throat were all examined bacteriologically.

In regard to the streptococci present, it appears to be as yet impossible to state whether those found are pathogenic or non-pathogenic, the known methods of differentiation being as yet insufficient to show with certainty which is the one present.

In Lennox Brown's text-book it is stated that where the streptococci predominate, the constitutional symptoms may be more severe and sometimes be accompanied by an erythematous rash and endocarditis - not necessarily rheumatic and that such general symptoms are more marked in lacunar than in peritonsillitis or parenchymatous tonsillitis. He says that the endocarditis is not necessarily rheumatic which may imply that there is no actual joint affection.

In those cases of tonsillitis which have much local

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* Hare's text-book of medicine states that acute tonsillitis is sometimes caused by a streptococcus and sometimes the organism of rheumatism of Poynton and Payne is present and sometimes the diphtheria bacillus but in another paragraph he rather minimizes the association of sore throat and rheumatism by stating that in his opinion it is often only an indefinite form of arthritis.

reaction and go on to suppuration it is the staphylococcus group which seems to be the chief element present, and this suppurative result corresponds to what are known as the chief characteristics of staphylococcal infection and a case of acute quinsy which bursts and discharges itself freely is usually followed by a very speedy improvement, whereas many of the cases in this epidemic had a very protracted period of weakness and anaemia resulting.

It is, therefore, evident in my opinion that there was here present a series of sore throats, all with many characteristics more or less resembling one another but yet which could clinically be divided into two main types: 1st a group in which the throat symptoms were the most important factor in the case and which did not cause so much systemic disturbance, and 2nd a group in which the throat symptoms were practically to all appearance trivial but the general systemic infection was very marked. There was, however, no real boundary line between them and the special characteristics in all likelihood depended on the general health of the patient and of the special prevalence of the one or other groups of micro-organisms.

Now as to the bacteriology of acute rheumatism, probably the most noteworthy authorities at present are Poynton and Paine and on reference to Dr. Poynton's article on rheumatism in Green's Encyclopeodia, it is found that their opinion is that the disease will yet be shown to be the result of one and not several micro-organisms. The organism which they have isolated, the micrococcus rheumaticus, is not yet definitely decided as the essential cause. Muir and Ritchie state that it is best described as a streptococcus growing in short chains. Poynton and Paine state that in liquid media it grows as a diplococcus. Diplococci have been described as the cause by Triboulet and Coyon and Wassermann and others all more or less alike in their general characteristics. Singer on the other hand maintains it is an attenuated pyaemia due to infection with various pus forming cocci, but I think that the fact of there being no pus found in almost any case of acute rheumatism is rather against this view and the lesions are not such as are the result of staphylococci usually.

Andrewes declares that the micrococcus rheumaticus of Poynton & Paine cannot be differentiated from and is practically identical with the streptococcus faecalis.

Beattie finds that the organism of rheumatism gives various reactions in growth and infective powers which are not produced by the ordinary streptococci.

It is now quite recognized, however, that streptococci are very liable to alter their characters according to their surroundings. The association with other microbes, the change of nutrient media &c. make quite distinct alterations on any growth, thus it is now allowed that the streptococcus erysipelatus is the same as the streptococcus pyogenes. The streptococcus scarlatinae of Dr. Klein and the Streptococcus conglomeratus of Kurth isolated from sore throats are identical. Kurth remarks that this streptococcus is invariably present in such cases often pure and always predominant and is specially grouped as to indicate a high degree of virulence and these again, they state, are the same as the streptococci found in the ulcers and milk from cows' teats and udders when diseased.

Professor Glaister in his book on Public Health describes (in 1889) a streptococcus found on teats and in the milk of cows with diseased udder as micrococci in short chains, which although not intended as such is the description of the micrococcus rheumaticus. He considered these the same as the organisms in the Hendon case and that they were the cause of various scarlet fever cases.

It is now acknowledged that in the "Hendon" case there was a source of human infection not discovered till after the outbreak had been reported on by the

eminent authorities in the case so that the sore teats and diseased udders had not in them the actual origin of the scarlet and that the disease produced by feeding the calves on the milk was really a septicaemia and this with or without malignant endocarditis so-called, may be but a very severe form of the effects of the rheumatic poison as stated by Poynton in the article on rheumatism referred to.

Not only so, but many leading authorities now state that there is no such disease as scarlet fever in cattle and that therefore the organism which they described as the streptococcus scarlatinae or conglomeratus was not the only factor in these cases.

SIMILAR OUTBREAKS OF SORE THROAT FROM MILK-BORNE INFECTION.

Most of these are referred to in Swinthinbank and Newman's Bacteriology of Milk.

(1) At South Kensington (1) in 1875 twenty persons who had used cream from a district where 119 cases of sore throat had occurred, some of them had scarlet fever.

(2) At Oxford in 1882 (2) fourteen cases sore throat and six of scarlet and one of diphtheria. There was a case of scarlet fever at the dairy for a week and the cases developed throughout the following week.

(3) At Upton and Macclesfield (3) in 1889 there were eighty-three cases of sore throat, thirty eight of scarlet fever and two of diphtheria, all using milk from one supply.

(4) At Brighton (1901) nine cases of sore throat and seven of scarlet all considered by Newsholme due to milk infection, indirectly through one girl partaking of milk from dairy with human infection.

(5) At Glasgow 1889, Prof. Glaister investigated epidemic; found ulcers on teats of two cows and both were casting hair and epidermis copiously and a calf fed on the milk developed similar signs and yielded streptococci from its blood, and he considered it a repetition of the Hendon Disease.

The symptoms were indistinguishable from scarlet

fever of a mild form, a particular streptococcus was isolated from the cow's eruptions and was regarded as the streptococcus anginosus (Andrewes and Hodder) or the S. Mastitidis (Savage).

The disease was mostly limited to the teats but in some cases the udders were also affected. There was no evidence of loss of hair in patches and no sores on the hands of milkers who declared there had never been any ropy milk.

Those using this milk pasteurized were not affected.

(6) In London and Surrey, &c., 1909, Sir Shirley Murphy reports an outbreak very widespread (over 400 persons affected) but traced ultimately to one supply, where it was found that most of the cows had scabs on the teats and a newly calved heifer was specially bad. This heifer calved on the 24th May; her calf died about four days afterwards, i.e., about the 28th May, and the milk was first used about the 7th June, which was the date on which the first cases of the outbreak were found. Two cases of supposed German measles proved to be scarlet fever and three cases of sore throat were also found at this dairy, but these had developed at the same time as the Surrey cases.

The foregoing have been all examples of a scarlatinal infection as well, - in fact, mostly scarlet fever.

Other outbreaks in which the scarlet fever element is practically absent are recorded:-

(7) At Aberdeen 1881, 300 cases of sore throat in 90 families out of 110 supplied by one dairy. Three deaths occurred in old persons and beyond the fact that there was no false membrane it was similar to this.

(8) At Rugby in 1881, Dr. Geo. Wilson reports about 90 cases in one school, and infection in 15 out of 37 houses supplied.by the same dairy in the town. A gargety cow was suspected as the cause.

(9) At Dover in 1884, 188 persons affected in four days, in the best residential districts several cases in many houses, vesicular eruption of the throat and very much enlarged glands were prominent features, and slow recovery was the rule. Among the sequelae were erysipelas, rheumatism and general septic conditions. In this epidemic some houses with a double milk supply showed those using the other milk free from the epidemic, whereas those using the suspected milk were frequently affected.

The milk was stopped and sent elsewhere and disease followed it, supposed to be due to aphthous disease in cows. Practitioner 1884, Vol. 1. p. 467.

(10) At Edinburgh, 1888, in an institute (Sims Woodhead and J.M. Cotterill) cowpox was supposed to be the cause. Sixty cases between 10th and 20th, milk stopped till November 7th, and in five days 25 more cases occurred,

then milk was boiled and there were no fresh cases.

(11) At Craigmore, Rothesay in 1890, 80 cases were recorded between 16th March and 2nd April. Three children died and there were many severe cases of erysipelas; two of the milkmaids had sore throats between 11th and 17th March.

On April 2nd milk was stopped, and the epidemic ceased. It was used again on May 6th and by May 10th fresh cases developed.

(12) At Hackney 1900 (J.K. Warry), 151 cases of septic sore throat in 88 houses. The tonsillitis is here said to have been not follicular, temperature was high and prostration great, and convalescence protracted. One death from pneumonia with septicaemia and two cases of acute nephritis. Many children affected, temperature remittent and persisted after tonsillitis desappeared lasting for nearly two weeks, and there was profuse sweating.

One case of purpura haemorrhagica.

Those using boiled milk escaped.

Bacteriology of milk failed to show cause.

Cause unknown.

(13) At Lincoln in 1902, Dr. Brook, 75 cases in the month of May in one week. Erythema of face and furred tonsils. 2/3rds of cases had scarlatinal rash and

desquamation was found in 1/3rd. Joint affections and peritonitis as sequelae. Cause unknown. Cases mostly slight. No infection was communicated to others. Nearly all were adults. Those who used boiled milk escaped. An organism was separated from the throats by Dr. Klein.

(14) At Bedford (1902), 42 cases in 22 families; symptoms like influenza at first and gastric disturbance twice as many females as males and 22 were under 20 years of age.

Bedford (1902) continued; those using boiled milk escaped, many using cream and fruit on a Sunday were specially bad, and in some families the disease was confined to those using cream. Milk examined a week later showed no evidence of the infection, but the dairyman acknowledged that his only precaution against infection from milk from diseased udders was to pass the milk through the separator which he thought destroyed the infection.

(15) At Woking (1903) 250 cases and eight deaths occurred. In addition to all the complications in the Cambuslang epidemic there were erysipelas and peritonitis. Adults were chiefly and most severely affected. One milk supply had four cows yielding pus from certain teats. The farmer himself had had sore throat followed by joint infection in September and when

the epidemic occurred in October his wife and four children were also attacked.

(16) At Belvedere Hospital, Glasgow (1904), 37 cases of sore throat occurred after a new cow was added to the dairy supplying the milk. This cow had udder disease, and within a fortnight 1/3rd of the cows had sore teats, - the hands of four milkers being also affected with sores.

(17) At Paisley (1904 by Dr. Robb). About 100 cases from milk of one farm where cows had recently had cowpox.

(18) At Colchester (1905) 140 cases in residential part of the town, many servants attacked, large proportion of adult females. Incubation period seemed to be about two days and cases were rather severe; very few secondary cases; at the same time 60 cases occurred in the Army Barracks which received milk from the same farm, and on this farm a cow was found yielding pus from a ~~diseased~~ eased udder, and six cases of sore throat occurred at this farm during the epidemic.

(19) At Boston, U.S.A. (C.E. Winslow, May 1911), a very wide outbreak occurred, about 1400 cases in Boston and neighbouring districts; reported in the Journal of Infectious diseases, Chicago, January 1912, said by the writer to be the first outbreak recorded in America. The description is almost the same as in the Cambuslang epidemic, but there was a greater variety of complications and several deaths mostly from septicaemia and

peritonitis. The description of the tonsillar condition corresponds very closely to that given in this paper.

Notice is made that throat and ear affections were very rare and the same remark applies to this epidemic. Abscesses of cervical glands were not uncommon, the deeper tissues being much affected and remaining painful for a long time. With children and young people the whole course of the disease was light, with the old or those with low resistance it was most severe, and almost any weak organ might be affected. Rheumatism was the most frequent complication. Erysipelas was very common, and there were also found pleurisy, pneumonia and heart affections. One lady had sore throat, erysipelas, pneumonia and pericarditis. In very severe cases, and probably all the deaths, septicaemia or peritonitis was present.

Throat examination showed no constant organism, but four cultures from internal organs showed streptococci, in each case, all apparently the same kind of organism, according to Prof. Theobald Smith Howard, Medical School.

There was a slight increase in the average number of sore throats in the distridt on May 4th, then marked increase from May 9th, culminating in a very widespread outbreak on 14th and 15th, and by the 23rd new cases ceased to appear. Practically no secondary cases were found, but 2, 3, 4, or more cases were commonly found in one

household more women than men were affected, few children and almost no infants were affected. Young adults (16-45 years) were the most frequent sufferers. There were 37 fatalities, all in persons over 65 years, and of those affected after the age of 75, 50% died.

The dairies supplying milk were not examined till after the 15th of May and then no evidence of disease was found to associate the outbreak with disease of cow's udder. There was a regular inspection usually carried out in connection with this milk supply by a veterinary surgeon, who tested the milk for bacteria per cc., and although the figures would not lead him to suspect any of the milk, yet I think the comparative figures are probably of importance. His reports for the month of May show in the suspected milk, Bacteria per c.c. of milk mixed.

May 1st	200,000	120,000	140,000	in various samples.
" 3rd	23,000	75,000	250,000	
" 5th	30,000	15,000	10,000	
" 8th	35,000			
" 10th	170,000	200,000	80,000	
" 15th	45,000	70,000	75,000	
" 18th	100,000	130,000	60,000	
" 24th	150,000	150,000	130,000	
" 26th	45,000	300,000	200,000	
" 29th	85,000	95,000	45,000	

Only 7 separate samples showed over 500,000
22 " " " 250,000 to 500,000
49 " " 100,000 to 250,000
504 " " less than 100,000
882 " " " 50,000

Now although none of these are in themselves by any means suspicious, yet on the first of May there seems to

have been a moderate number of bacteria present compared to the following days until the 10th May and the number of cases taken ill corresponded slightly to the time of these increases, thus on the 4th May there was a slight increase and then on the 14th a very great increase, which would allow about 3 days as an incubation period.

Dr. Winslow's conclusion, however, was in favour of human infection as there had been sore throats in the district where the farm was, for a time previous. He says:- "The lesson to be drawn from the outbreak is that even a most carefully supervised milk supply is open to the danger of grave infection from carrier or unrecognised case of disease. The only real safeguard against such catastrophies lies in pasteurization carried out by the holding system and preferably in the final packages."

On a consideration of these twenty outbreaks, nineteen previously reported and this one, they divide themselves into two main groups, one group including Nos. 1-6. Scarlet fever was the chief disease, or was frequently present.

In the Journal of Hygiene, Vol. ii. p. 150., Newsholme states that scarlet fever may be caused by infected milk, containing the contagium in such an attenuated form or minute quantity that no symptoms manifest themselves except as anomalous sore throat with fever.

I have frequently found that in an outbreak of scarlet fever (and it is an acknowledged fact) some members of a family will have a sore throat practically identical to the others who go through the full course of scarlet fever as if the same infection occurred but with a less result; where I have seen it most frequently is in the mother of children with fever or in those who have previously had the disease.

The special organism of scarlet fever cannot be said to be definitely settled. No doubt a streptococcus has been described as the essential cause and can always be found in the throat, but that organism has been considered by Klein and Kurth to be the same as found on the teats and in the milk from diseased udders. In cases (1), (5) and (6), by eminent authorities, this microbe was considered to be the cause of the scarlet feverand in (1) and (5) it was said to be the 'Hendon' disease, but No. 1, the noted case , so often regarded as proof of the possibility of scarlet fever in cows and calves is discredited by many eminent veterinary surgeons who declare that cows cannot take scarlet fever, and that a human source of infection did exist. It is not denied that they may take an acute mammitis with much of the general systemic infection and very little evidence of the disease in the udder, in fact, the most infectious and worst cases give little signs at

all, even the milk being but slightly affected beyond diminution in quantity. Is it not possible, (I think it very likely) that these cases are much more after the nature of the erysipelas and are due to an almost pure streptococcal infection, whereas the others with much hardening and suppuration are the result of a mixed infection from both staphylococci and streptococci, or it may even be that the localized abscess is more or less almost entirely staphylococcal and less virulent for the human species.

Prof. Glaister's description of the organism found in No. 5 is as I have said, just what would be given of the micrococcus rheumaticus. Again in No. 6 the fact that there were two cases supposed to be German measles, and then verified as scarlet fever is very suspicious that there was here also a human source of infection.

Nos. 2 and 4 had distinct evidence of contact with infectious person and in No. 3 no cause was discovered.

From another point of view I think that these scarlet fever outbreaks were more than infection from the cow. The majority of the diseases caused by the ordinary streptococci as erysipelas endocarditis, &c., tend rather to predispose to a second attack, whereas scarlet fever does the opposite. During the epidemic at Cambuslang there were three cases of scarlet fever notified to the medical officer. I reported two of

them, but as I have said, there was a possible source of human infection for these over and above this milk epidemic, but another case showing, I think, that the scarlet fever and the sore throat epidemic were distinct, was in a family where a boy had the sore throat followed by endocarditis and as the sore throat was very like scarlet fever it was supposed at first that he was the source of infection of two children who took scarlet fever, just as he was recovering. He showed, however, no desquamation at this time and a few days after the others were removed to hospital he developed distinct scarlet fever running quite a normal course.

In my opinion it will be found that all, or practically all, epidemics of scarlet fever spread by milk arise from infection from a previous case of scarlet fever amongst those who have to do with the milk. It has been so in all the small outbreaks of which I have had experience.

The other outbreaks are all more or less similar to that forming the subject of this paper, but that at Lincoln (No. 13) is very suspicious of a scarlet fever element being also present.

The clinical features were very much alike, but in No. 1 it is stated there was no false membrane in the throat, and in No. 12 that the tonsillitis was not follicular.

Then those affected seem to have been in the majority of cases just those specially liable to sore throats, i.e., from 15 to 45 years of age, but in one epidemic it is stated that the proportion of children was large. It is worthy of note, however, that infants and very young children seem to have escaped, apparently they were insusceptible to the infection or the result was not similar to that in those of less tender years. Now in this respect it may be worth while making comparison with acute mastitis in women. This is not uncommon, and in my opinion closely resembles the disease in cows, and I think that it will be admitted that here there are found varieties of acute mastitis. In some cases there is a local nodule in the breast moderately hard and circumscribed which goes on to abscess formation and soon discharges pus. In others there is found a red mark extending from the nipple with an appearance similar to that of a lymphangitis and probably with comparatively little hardening, but with considerable fever. Most medical men recommend that the infant should not be suckled at an inflamed breast, but many mothers consider the frequent feeding, i.e., emptying of the breast, to be the best treatment for the condition, and I must say that beyond what appeared to be a little sickness I never saw much harm arise; as I have already said, it is the usual practice where calves are kept to give them the milk from

the diseased udder if they are then getting milk at any fate; it is the usual result to find no ill effects.

Of course, in these cases the milk is usually mixed; sometimes boiling water is added and they may not be getting sufficient of the infection, whereas the calves fed with the milk from infectious udders by Dr. Klein and Sir Wm. Power in their experiments may have received a very large supply of the infectious material, and the natural forces giving ordinary immunity may have been insufficient to withstand the excessive infection and the germs may thus have become effective in producing disease not really scarlet fever but streptococcal septicaemia; in ordinary circumstances the young, both in cattle and in the human subject, are more or less insusceptible to this form of streptococcal infection, and H. Tilley states that sore throat rarely occurs in children under 4 years.

In No. 12 it is reported that the fever remained up for nearly a fortnight after the throat symptoms had subsided. In the Cambuslang district there were several cases similar, although I did not find it remain so long elevated. I can only account for it in the theory that there was still some systemic infection in the blood as in every other way the patient seemed normal, even as to his general feelings and as to the actual pulse rate and all local evidences of disease. It may

be that this continued elevation of temperature was more general than has been recorded, as the patients felt perfectly well, and unless a regular temperature chart was being kept it was practically certain to escape notice.

In practically every one of these epidemics where a cause seems to have been attributed there is the history of inflamed udder or sore teats, or both, and in no case has it been shown definitely (in three it is suspected) that there was any likelihood of a human source of infection.

In one (No. 15) of the suspected human sources there is also distinct evidence of udder disease being present as well, and I think it is much more likely that the outbreak resulted from the four diseased cows than from the farmer whose throat had been ill a month previously, and was followed by joint affection because his infectiousness would have ceased by that time, and if he had been at all infectious his wife was likely to have been infected then. In another (No. 11) the Rothesay outbreak, there were apparently two milkmaids who might be the cause, but I cannot believe that they could be the cause when the use of the milk was resumed on May 6th, seeing their throats were sore only from the 11th to 17th March, and in all likelihood they were rather the first sufferers from the infection from some of the cattle. In the Boston epidemic (No. 19) I have suggested that even there the

bacteriological records may mean udder disease.

For the others in which no cause can be found, but which were undoubtedly milk-borne, it is quite a legitimate inference that disease may have been present, although unknown to the milkers. The non-contagious nature of practically every outbreak, that is, from one person to another, seems to be likewise rather in favour of this theory and against that of human infection being transmitted to the milk.

I am afraid, however, there is another side to the question. If this is the case that these weeds in cows are apt to cause an outbreak of sore throat, why are there not continually outbreaks occurring? There are few large dairies and not many small ones, where weeds are not to be found very frequently, and therefore there is scarcely a community where sore throats should not be more or less apparently endemic.

Although I believe that small epidemics are really more frequent, than is usually recognised,-I have often remarked how often quite a number of sore throats occur more or less simultaneously, - yet they are in a way so unimportant that no notice is taken of them, and there is further the belief that they mostly arise from cold or wet.

Another reason, however, is very likely, although not yet proved, streptococci vary enormously in their

virulence under different conditions, and again, abscesses of the udder, or rather it may be more comprehensive to say acute infectious disease of the udder varies likewise, not only in virulence of organism but also in the nature of the organism, i.e., staphylococcal and streptococcal, and that variation and combination may make a great difference in the virulence towards the human species. I have said that the M.O.H. found staphylococcus aureus in every swab examined, but his examination was not exhaustive as the special point with him was that there were no diphtheria bacilli. The lesions, however, in the internal organs showed in the Boston outbreak a streptococcus and there was no reason to doubt the same was present here, so that there was no doubt of the mixed infection which may have a considerable bearing on the virulence, and may account for some outbreaks being very widespread and serious, while others are very mild.

A word may be said in reference to the variety of sequelae which followed the inflamed throat.

1st, a continued temperature after all throat symptoms had disappeared, i.e, the infection had become systemic, this was noticed very markedly in the Hackney outbreak, and also in the Cambuslang outbreak.

2nd. Acute rheumatism; this has been very frequently noted, in some cases only as rheumatics but in many outbreaks as perfectly distinct acute rheumatic

fever. The incubation of this was usually about nine days after the throat commenced. Now the disease is considered to be due to the micrococcus rheumaticus which by some is described as a streptococcus, and the description is very like that found in describing the organism in some cases of acute mamitis. From the direct source of the infection in these outbreaks it is impossible to come to any other conclusion than that the microbe of this disease was introduced by the milk and that it must be the same microbe as is found in the diseased udder or was the same until it may have become altered in its passage through the tonsillar tissue and the blood. In corroboration of this I think the cases of rheumatic joints in cattle which I have narrated, and which were the direct result of the mammitis in the cows. Even although Beattie states that there are various characteristics in those microbes which he describes as being quite different from the usual streptococci, it may still be possible that the germs assumed those characteristics only after developing in the joints affected with rheumatism. I consider that we ought to consider that the microbe of rheumatism is the same as the streptococcus of inflamed udders.

3. Heart affections, endocarditis and pericarditis; these have long been associated with rheumatism and in these epidemics we have ample verification of the fact that it is in children that heart affections are most liable to

develop, and that adults may go through a very severe attack of rheumatic fever without the slightest evidence of valvular disease, but that even in adults both endocarditis and pericarditis may occur.

The remarks as to the bacteriology of the acute rheumatism apply here.

4th. Lung conditions, pleurisy and pneumonia; these have been often reported, but as at the time of the outbreak I did not think of these as associated I cannot remember anything of the frequency with which they occurred. As I write this paper, however, I have a patient, a boy about 12 years of age who had had sore throat for fully a week about as typical, red inflamed sore throat (rheumatic) with only streaks of whitish mucous about it, as it can be and who has now developed distinct pleurisy over the right lung. This case, although not in the epidemic shows the close association of the rheumatic sore throat and the pleurisy.

One thing I remember about the pneumonia cases was that on several occasions sore throat and pneumonia did occur and my assistant said that I should call these, ~~and~~ milk cases also, rather in sarcasm for me blaming too much on the milk.

5th. Skin conditions, erythema nodosum was the most frequent complication and it has long been associated with rheumatism and is considered to be microbic

erysipelas is undoubtedly so.

Purpura is likewise often associated with rheumatism and it has been reported in several of the epidemics.

6. Peritonitis. I saw none but three epidemics give such.

7. Septicaemia which seems to have been the cause of many of the deaths. I saw no cases which I could designate as such, but it is in all likelihood only an aggravation of the condition I observed, and which has been reported also in No. 12 of continued rise in temperature even after throat symptoms disappeared. A case I had just shortly after this outbreak was a very good example of a similar condition; a young man, 28 years, a gardener blistered his heel with a new boot. He went about and it became acutely inflamed (his brother was beside him with erysipelas of the face) then an acute inflammatory swelling started just to the inner side of the front of the thigh about a hand's breadth above the knee. This suppurated and I opened it, and apparently he was relieved for two days, then began to suffer from headache and vomiting and purging.

This continued for about a week and the man was exceedingly ill with a very rapid pulse, but his temperature scarcely ever rose above 101°F. A slight rheumatic swelling of both wrists developed and was relieved by aspirin, then the left shoulder and arm became ill and

were likewise relieved by aspirin. His pulse, however, was so very rapid and feeble, about 120-140 usually, I did not push the aspirin but allowed him to take just so much as to keep down the pain. For fully a month this man lay with a temperature of about 100° and a pulse seldom under 110 per minute, but with not a single lesion otherwise to account for it. The abscess of the leg healed up normally, and also the heel. In three months he was able to resume his work. I mention this case as showing a relation between a rheumatic condition and a septicaemic condition.

8th. Various other conditions, chorea, ovaritis and apparently as some records have, almost any weak organ may be affected.

It seems therefore to be the conclusion that the same group of germs being taken into the system in different people may give a very different result in some from that in others, and that several diseases which appear more or less quite distinct are really different manifestations of the one disease arising from the different resisting powers of the various structures along with a difference in the amount of the infection which has been received.

PREVENTION OF SUCH EPIDEMICS.

In quite a few of these outbreaks it has been clearly demonstrated that milk infected but boiled or pasteurized did not give the infection to those partaking of it. It appears, therefore, a very simple matter to entirely prevent these outbreaks, either as is suggested in the Boston outbreak by pasteurizing all the milk especially in the final packages in which delivery is made to each customer, or by each household pasteurizing or boiling its own supply.

Boiling is the safer and surer of the two methods ~~and~~ but a great many people will not take boiled milk owing to the taste of it. The question also arises, is boiled milk as nourishing and satisfactory as a food as fresh unboiled milk? I think there is no denying the fact that besides being much less palatable the milk is also less valuable as a food.

Vincent states:- "The value of milk is that it is a raw food. In all circumstances the cooking of milk for the use of infants gravely impairs its nutritive value. No serious amelioration of the mortality from zymotic enteritis can be anticipated until the fullest protection for pure raw milk is secured for every infant, i.e., the milk shall be preserved fresh and unboiled."

My experience leads me to the same conclusion and I think that even for children with summer diarrhoea as it is called there is no better treatment than sweet

milk as the food, but it must be sweet milk and not such as sours and becomes bad within a few hours of being delivered. Of course, I usually stop all milk and practically everything except albumen water for thirty six hours to commence the treatment. Besides there seems to be something in sweet milk which acts as a special digestive aid and which has an effect on the whole nutritive mechanism. Cases of scurvy have been recorded in children fed only with boiled milk, and I have seen several children who were not actually suffering from scurvy but showed a tendency thereto becoming quite healthy when put on fresh cow's milk. For adults there may be little to be said about boiling the milk beyond the loss of some vital energy, but here the taste is often sufficient to keep one from using boiled milk.

The difficulty was thought to be got over by pasteurizing the milk and this seems also in all cases to have been effectual or nearly so in preventing the infecting milk doing any harm, but even pasteurization seems to diminish the nutritive value of the milk and another objection arises which I think ought to be put on record, and that is that children fed entirely on pasteurized milk are very liable to become victims of tuberculosis.

This was my experience in regard to the use of pasteurized milk in Glasgow supplied through the

Corporation depots in bottles with comparatively small quantities in each so that no milk need be exposed for any length of time. Now those using such milk are naturally those who take a very great interest in their children's health and with whom one would expect to find comparatively less illness than usual but the number of those whom I had to attend for tubercular adenitis of neck, tubercular peritonitis and tubercular meningitis was excessive. I have no direct information from the Glasgow Public Health Authorities why they discontinued the use of this pasteurized milk, but a medical practitioner in Glasgow stated to me that it was because the children did not continue to do well even although they seemed to do all right and to be mostly free from attacks of diarrhoea for some months.

It is my opinion therefore that neither the boiling nor the pasteurization is a satisfactory method of treating milk, and that although they could be utilized on occasions when infection was suspected without any special disadvantage to health yet the infection is almost invariably widely spread before it can be known that it is present owing to the fact that most outbreaks require a few days to develop the disease in sufficient numbers to warrant a conclusion as to where it originated.

Another method is the use of preservatives in milk

and although many of these will undoubtedly keep the milk sweet and prevent the rapid growth of the infection in the milk yet they cannot be used in sufficient quantity in the milk to actually destroy the micro-organism but in many cases make the organism assume forms which when freed from the preservative and placed on suitable soil grows most luxuriantly. It is not a safe method to adopt and may be the means of allowing milk to be sold which would, without the preservative, become rancid so quickly that its use would not be possible.

It seems to me, therefore, to be necessary to go to the origin and prevent, if possible, the infection getting into the milk and this can only be done by great care being exercised by all who are concerned in the milking of the cows and distributing the milk.

Although I believe that most of these epidemids of sore throat and rheumatism arise from the mammitis, yet there are also the other infections such as scarlet fever arising from human source and the milk requires to be protected from both of these.

Milk is so general and so important an article of diet that it ought to be made compulsory that everyone suffering from any disease which may give infection per the milk or nursing or attending to any infectious disease should be rigidly excluded from either milking the cows or assisting in the storing or distribution of

the milk. Also that all milkers should be required to wash the hands carefully before milking; that it should be done after every cow may not be necessary, in fact some milk experts prefer to keep the hands perfectly dry all the time of milking, but I think that the water supply should be so arranged that a tap of running water is available in every byre or close to it, at which the milkers can easily wash the hands in running water if they have become soiled by contact with a diseased udder or in any other way. It is a much more likely method of having infective material washed off the hands than that of using a vessel of water with some antiseptic in it, as is the usual custom in some dairies.

Of course if one took fresh antiseptic and fresh water each time it would be all right, but that is impracticable, whereas to wash at a tap of running water is very simple and effective; and the water is always fresh and free from pathogenic germs, or ought to be.

Again there is the cow's udder to be considered, it has been shown that washing the udders does not give so good results as simply rubbing off all the dirt with a dry cloth and milking with udder and teats dry. The cow's bed should be kept as dry and clean as possible, and although the germ theory undoubtedly holds true so does the old-fashioned one of a 'chill' and the housing and care of the cattle should be such as will reduce

the chances of undue exposure or imperfect ventilation acting as the exciting element to the weed.

All utensils must be carefully washed and scalded regularly but most dairies already do this very thoroughly.

Then in regard to the actual 'weed' or 'garget' or acute chill even before the udder condition is recognisable, the precaution ought to be taken of withholding that milk from sale. If the farmer considers it fit for human food let him keep it and feed himself and his servants on it, the latter will soon object and he himself is not likely to use much. I believe, however, that it may safely be used for feeding calves and even in the manufacture of butter as the bacteria of lactic acid fermentation seem to be able to destroy pathogenic germs - at least the latter appear to die before either the butter or the butter-milk is ready for use.

I think we ought to go further and prohibit milk being sold which was milked from an animal suffering from any disease whatever. Such milk may be used for cheese or butter as I have suggested and as this would penalize the farmer to the advantage of the general public then it may be justly advocated that the farmer should receive compensation for the loss of sale on the milk. That ought to be allowed as is done at the Boston supply dairies to anyone who notifies that disease is present in the dairy, but where this is only discovered by others after the

infection has been spread, then let no compensation be allowed.

Another precaution which ought to be taken is that all the healthy cows ought to be milked first and then those which are suffering from disease; by this method there would be little or no fear of carrying infection on the hands directly from a diseased cow to a healthy one or even of the milk from a healthy cow coming in contact with a hand covered with the discharge from a diseased udder.

If all those concerned with the supplying of milk were aware of the dangers of impure milk and of the penalties which could be imposed for the supply of milk which is deleterious to the health of the consumer as well as of the fact that allowance would be made from the public purse if they refrained from selling any suspected milk, and notified the authorities of the fact so that investigation could at once be made and directions given as to the method to adopt in each case, then I think it is almost certain that there would be no such outbreaks as are usually recorded, but as I have already shown there are a few cases in which no cause can be found, although I think these would then be very much fewer because it would be to the advantage of the farmer to be careful and to refrain from selling suspicious milk, rather than to conceal a case that outsiders were unable to detect without very

great trouble.

Briefly then my conclusions from a study of this epidemic and of comparison with previously reported epidemics are:-

1. Epidemics of sore throats due to milk-borne infection may arise from milk infected from a human source or from a bovine source, i.e., disease of the udder chiefly acute mammitis.

2. Those epidemics associated with scarlet fever are almost entirely (if not altogether) due to infection from a previous case of scarlet fever in someone concerned with the dairy, whereas those associated with acute rheumatism and other allied affections are from bovine source.

3. The sore throats in the latter class are due to such organisms as are usually found in the milk from diseased udders and in the ulcers on the teats, especially the staphylococcus pyogenes aureus and the streptococcus pyogenes in one of its numerous forms.

4. The organism associated with acute rheumatism and with its allied conditions must be the same organism as is present in these sore throats and therefore in the acute mammitis, although it may have become modified in its characters owing to the difference in its mode and place of growth.

5. The infective virulence of all cases of acute mammitis is not the same, but seems to be modified by the special nature of the inflammation present and of the relative numbers of the different varieties of organisms present.

6. The tonsillitis most likely to be followed by acute rheumatism and other general infection is that whose symptoms are a diffuse redness of the fauces, tonsils, soft palate and uvula with comparatively little swelling of the tonsils, and with little exudation about the tonsils.

7. The tonsillitis showing much furring and swelling of the tonsils is most likely to result in peritonsillar abscess or abscess of the lymphatic glands of the neck, and comparatively little general systemic disease.

8. There is no distinct boundary line between these two varieties and most commonly there is a combination of both.

9. There seems to be an incubation period of one to two weeks after the commencement of the throat illness before the rheumatic condition develops and even after this period some who have apparently escaped may develop the acute rheumatic condition if exposed to cold or wet or other conditions favourable to its development.

10. By encouraging and insisting on those concerned with the supply of milk being careful and using such

precautions as I have recommended these epidemics should almost entirely, if not altogether, disappear.